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LETTERS TO THE EDITOR

Subaortic Obstruction After Pulmonary Artery Banding in Patients With Tricuspid Atresia and Double-Inlet Left Ventricle and Ventriculoarterial Discordance

The article by Franklin and associates (1) and the accompanying editorial by Puga (2) raise several important issues that deserve further comment.

Does pulmonary artery banding produce subaortic stenosis? The premise of both papers (1,2) was that pulmonary artery banding exacerbates or promotes development of subaortic stenosis in infants with tricuspid atresia and double-inlet left ventricle with a discordant ventriculoarterial connection. This premise, in my opinion, is erroneous and is without proof.

We have carefully examined the natural history of the ventricular septal defect in tricuspid atresia (3–9) and concluded that there is a great natural tendency for the defect to close spontaneously whether it is an isolated defect or part of a more complex cardiac anomaly such as tricuspid atresia. We further observed that the incidence of spontaneous closure in tricuspid atresia is similar to that found in isolated ventricular septal defects. We have also noted that pulmonary artery banding is not causally related to ventricular septal defect closure and cited reasons for such conclusions (6,7,9). 1) Newborn infants were found to have a constricted ventricular septal defect (10,11), and 2) narrowed defects were reported without previous pulmonary artery banding (6,11,12). We have surmised that if banding had not been performed, the infants would have died from severe congestive heart failure and, therefore, the expression of natural closure of the defect would not have been possible. Pulmonary artery banding improves patient survival, thus giving an opportunity for expression of the natural history of the ventricular septal defect, which is spontaneous closure.

In the study of Franklin et al. (1), there were 33 patients without subaortic or aortic arch obstruction who underwent pulmonary artery banding. During a mean follow-up period of 8.5 years, 5 (22%) of 26 patients developed subaortic stenosis (ventricular septal defect closure). This 22% incidence of defect closure is not higher than that expected for isolated ventricular septal defects (13–15) or for those associated with tricuspid atresia (7–9). Therefore, it is difficult to blame pulmonary artery banding as the cause of closure of the defect. Furthermore, when Franklin et al. (1) examined the ratio of ventricular septal defect to ascending aorta diameter, they found that subaortic stenosis developed in a group of patients whose ratio (0.6 ± 0.08) was much smaller than that in the group with a larger ratio (1.03 ± 0.15, $p < 0.001$). The ratio between the aorta and the pulmonary artery, when similarly examined, did not discriminate between the two groups. These data indicate that it is the size of the ventricular septal defect at the time of initial presentation, and not pulmonary artery banding, that is a predictor of future, significant subaortic stenosis. In a recent abstract presentation, Matitiau et al. (16) examined the two-dimensional echocardiographic area, adjusted to body surface area, of the ventricular septal defect and found that infants who developed subaortic obstruction had a significantly smaller ($p < 0.001$) defect than did those who did not.

Thus there is overwhelming evidence that it is the intrinsic nature of the ventricular septal defect rather than pulmonary artery banding that is responsible for spontaneous defect closure. The pulmonary artery banding, by preventing premature death, allows expression of the natural history of spontaneous closure of the defect. However, I agree with the thesis of Freedom et al. (17–19) that myocardial hypertrophy develops after pulmonary artery banding, which in turn is a risk factor for poor prognosis after the Fontan operation (20), and that alternative treatment approaches must be developed to circumvent the problems of initial palliation and subsequent "correction."

Norwood or Damus-Stansel-Kaye? Disconnecting the main pulmonary artery near its bifurcation and anastomosing the proximal end to the side of the ascending aorta for anatomic correction of transposition of the great arteries was initially reported by Stansel (21) but it appears to have been independently and simultaneously conceived by Damus (22), Alvarez Diaz et al. (23) and Kaye (24) and is commonly referred to as the Damus-Stansel-Kaye procedure (25,26). A similar operation utilized by Franklin et al. (1) was designated as a Norwood procedure. Although the semantics are of no concern (especially in view of the fact that Franklin et al. describe the surgical procedure in detail), I am of the opinion that when eponyms are used the term "Norwood procedure" should be used when a hypoplastic aorta is anastomosed to the main pulmonary artery after its transection. The term Damus-Stansel-Kaye or, simply, Stansel procedure should be used when the distal end of the transected pulmonary artery is anastomosed to a normal or nearly normal-sized ascending aorta, end to side.

Which operation to perform? The recommendations of Franklin et al. (1) that a main pulmonary artery to ascending aorta anastomosis, end to side along with a central aortopulmonary shunt for patients with subaortic obstruction at presentation and for patients identified as having a high risk for developing subaortic obstruction (ventricular septal defect/ascending aorta ratio <0.8), seem reasonable and should be followed. They also recommend pulmonary artery banding for patients without evidence for systemic outflow obstruction and then follow up the patients with frequent echocardiographic-Doppler studies to detect the onset of subaortic obstruction. By contrast, Puga (2) appears to recommend a more aggressive palliative intervention for all patients. The latter is probably not advisable until it is shown that the mortality rates with this aggressive palliative approach are brought down to those seen with isolated pulmonary artery banding.

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Reply

We are grateful to Rao for his interest in our data and endorsement of our suggestions. The main point is that it may be premature to discard banding of the pulmonary trunk as initial palliation for selected infants, provided that the aortic arch is not obstructed and the ventricular septal defect is of adequate size at presentation. We agree that using eponymous terms for operations may cause confusion which is why we described in detail the techniques of palliative surgery used in our patients.

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